

grossly different from that of albino hairless mice in structure and function, it would appear wise to follow the recommendations of the Ad Hoc Committee of the American Academy of Dermatology. These include minimizing sun exposure and avoiding use of retinoic acid in the presence of sunburn until fully recovered, as described in the package inserts for the drug. Patients with chronic sun exposure, genetic predisposition to sunlight-induced skin cancer or unusual susceptibility to sun-induced skin damage should be particularly cautious when using retinoic acid. I would reemphasize that at this time the data are incomplete and inconclusive, and may or may not apply to human use situations. However, they cannot be ignored and strict adherence to the recommendations noted above coupled with an awareness of new developments as they evolve will allow physicians maximum latitude in selecting therapy suitable to a patient's needs while vigilantly protecting the patient's best interest.

RONALD M. REISNER, MD

REFERENCES

- Hurwitz S: The combined effect of vitamin A acid and benzoyl peroxide in the treatment of acne. *Cutis* 17:585-590, Mar 1976
- Papa C: Scanning electron microscopic study of tretinoin (vitamin A acid) acne therapy. *Cutis* 17:575-580, Mar 1976
- Kligman AM, Fulton JE Jr, Plewig G: Topical vitamin A acid in acne vulgaris. *Arch Dermatol* 99:469-476, Apr 1969
- A Possible Influence of Topical Retinoic Acid on Sunlight Induced Cancer—Report of the Ad Hoc Committee of the American Academy of Dermatology. Evanston, IL, American Academy of Dermatology, Jul 1978
- Epstein JH: Retinoic acid and experimental carcinogenesis. Read before the Pacific Dermatologic Association, Coronado, CA, Sep 18, 1978

New Concepts in Pathogenesis of Acne

ACNE is a multifactorial disease in which hormonal, biochemical, microbiological and immunologic factors interplay. The most widely accepted theory on the pathogenesis of acne is the free fatty acid theory. According to it, the follicular bacteria, particularly *Propionibacterium acnes*, produce lipases which hydrolyse sebaceous tryglycerides to free fatty acids (FFA) which in turn produce inflammation.

Arguments against the role of FFA are that no significant differences are observed between patients with acne and controls in (1) the free fatty acid content of skin surface lipids, (2) the sensitivity of skin to irritant effects of free fatty acids, (3) density of bacteria in isolated sebaceous fol-

licles of acne prone areas of skin and (4) lipolytic activity of strains of *P. acnes*. Recently, it has been shown that FFA are not inflammatory when injected into normal human skin. The possible explanation is that the earlier studies injected nonphysiologic amounts that did not reflect *in vivo* levels.

Recent studies have found that follicular bacteria, though harmless, may act as primary inflammatory agents in closed comedones. The mechanism of action probably involves the production and accumulation of bacterial cytotoxins and cytotoxic agents, as well as direct inflammatory activity of bacteria introduced into the perifollicular dermis following the rupture of the comedonal wall. The successful use of antimicrobial therapy in the treatment of inflammatory acne substantiates their role as causative agents. The mechanisms whereby bacteria produce the inflammatory reaction remain controversial and unclear.

S. MADLI PUHVEL, PhD

REFERENCES

- Puhvel SM, Sakamoto M: A reevaluation of fatty acids as inflammatory agents in acne. *J Invest Dermatol* 68:93-97, Feb 1977
- Puhvel SM, Sakamoto M: An *in vivo* evaluation of the inflammatory effects of purified comedonal components in human skin. *J Invest Dermatol* 69:401-406, Oct 1977

Role of Hepatitis Virus in Gianotti-Crosti Syndrome

PAPULAR ACRODERMATITIS OF CHILDHOOD (PAC) or Gianotti-Crosti syndrome is a transient non-pruritic papular dermatitis manifesting as monomorphic flat erythematous or flesh colored lichenoid papules occasionally scaly, with a predilection for extensor surfaces. It has been reported from infancy to early teens but is commonly seen in two- to five-year-old children, with equal sex incidence. Associated features include lymphadenopathy, malaise, fever and diarrhea. The lesions disappear within six weeks but can persist longer. On microscopic studies mononuclear cells and histiocytes are seen in the reticular dermis and perivascular spaces.

Nearly all patients have acute hepatitis which is usually anicteric and indistinguishable from viral hepatitis. During the acute phase of the disease, all patients have hepatitis B surface antigen in the serum with elevated serum transaminases. During the convalescent phase, three

months later, patients are found to have anti-hepatitis B core antibody in their sera. Antibody responses to the hepatitis B core antigen and surface antigen are identical in patients with viral hepatitis and PAC. These findings have suggested the hypothesis that PAC may be a manifestation of the primary infection with hepatitis B virus. PAC can progress to chronic active liver and serologic abnormalities may persist for several months. However, the hepatitis B surface antigen or e antigen has not been isolated from the skin.

The infectivity rate is low. Hepatitis B virus antigen has been found in sera of siblings and parents of patients many months after exposure.

Patients with infantile papular acrodermatitis have clinical similarity but lack evidence of liver disease and lymphadenopathy.

MARK OESTREICHER, MD

REFERENCES

- Eiloart M: The Gianotti-Crosti syndrome: Report of 44 cases. *Br J Dermatol* 78:488-492, Aug-Sep 1966
- Gianotti F: Papular acrodermatitis of childhood—An Australia antigen disease. *Arch Dis Child* 48:794-799, Oct 1973
- Colombo M, Gerber MA, Vernace SJ, et al: Immune response to hepatitis B virus in children with papular acrodermatitis. *Gastroenterology* 73:1103-1106, Nov 1977

Topical Corticosteroid Therapy

TOPICAL GLUCOCORTICOSTEROIDS are among the most frequently prescribed topical agents for an inordinate variety of cutaneous conditions by physicians in practically every discipline of medicine. Their striking, beneficial effects are reflected by the proliferating array of creams, lotions, ointments and gels containing such agents that is available to physicians. The prolonged and injudicious use of such agents, however, may result in a variety of disturbing side effects. These include the development of rosacea, steroid acne, perioral dermatitis, telangiectasia, stress, atrophy, hypopigmentation, hirsutism and purpura, and enhancement of infections. These adverse effects can result from the use of any of the topical steroid preparations but have been most frequently encountered with the use of the more potent fluorinated glucocorticosteroids.

The strong topical formulations of glucocorticosteroids currently available and new more potent ones appearing on the horizon demand the intelligent use of these agents if these serious side effects are to be avoided. Stoughton has classified the currently available commercial topical glu-

cocorticosteroid preparations into six groups, in respect to their potency. The science of the use of topical glucocorticosteroids involves the selection of the correct strength corticosteroid in the proper formulation for the particular disease process and the particular area of the body involved. The most potent topical steroid preparations are used as a part of the initial therapy for recalcitrant types of dermatoses or when a physician wants to obtain a brisk response.

Once suppression has been achieved, these agents should be replaced by less potent steroid preparations for the purpose of tapering off the topical agents or obtaining the least potent agent which will still be effective therapeutically. They should not be used over a long period. All areas of the body do not absorb steroids to the same degree. The face, scrotum, penis, groin and perianal areas have unique absorptive properties which tend to predispose these areas to the development of undesirable reactions. Further, if steroids are applied under occlusive dressing, such as Saran Wrap, absorption may be enhanced a hundredfold.

Therapists must be constantly alert for the earliest signs of evolving side effects. Open-ended prescriptions for these agents should never be given.

Reflecting the current misuse of topical steroids is the increasing number of patients seen in dermatological offices with conditions resulting from the unskillful use of topical steroid preparations. As Stoughton has noted, "The uneducated and inexperienced have no place in the management of dermatoses with topical glucocorticosteroids."

VICTOR D. NEWCOMER, MD

REFERENCES

- Kligman AM, Leyden JJ: Adverse effects of fluorinated steroids applied to the face (Special Communications). *JAMA* 229:60-62, Jul 1, 1974
- Stoughton RB: A perspective of topical corticosteroid therapy, *In* Farber EM, Cox AJ (Eds): *Psoriasis Proceeding of the Second International Symposium*. New York, NY, Yorke Medical Books, Donnelley, Dun, Publishing Corp, 1977

Role of Antibody Titer in Pemphigus

PEMPHIGUS is a vesiculo-bullous autoimmune disease of the skin characterized by the presence of an antibody against the intercellular substance of the epidermis. The antibody is bound *in vivo* at the site of the pathologic condition and is present in the serum. It is composed of all four classes